The exponential curve relating the prevalence of coronary heart attacks to the cholesterol plasma levels makes the epidemiologist happy. Clinicians, however, are faced with actually treating sick men rather than dealing with "large numbers." Working in a hospital, we were interested more in the events occurring in people belonging to the "low" segment of the curve than to the "high" segment. Why did so many coronary events occur with "large numbers." Working in a hospital, practically treating sick men, it was easy to perform and reproduce, was introduced just at that time. We couldn't, however, readily acquire the necessary antisera. Gerhard Kostner from Graz kindly supplied us with the immune sera, so we could then start our work. We thought that the survivors of myocardial infarction would represent a good group to be tested; J.L. Goldstein and colleagues had observed a group of survivors of myocardial infarction to verify some clinical aspects of the lipid hypothesis. After we collected and analyzed our data, it appeared that apoB was the best discriminator between survivors and controls and that the ratio apoB/apoA-I had the best absolute discriminating power. Our work received nearly unanimous confirmation, including results from groups studying patients whose disease was coronarographically proven. Our work contributed to the understanding of the clinical role of apolipoproteins.

In the following years, A. Sniderman and colleagues described patients affected by ischemic heart disease (IHD) as characterized by an isolated increase of apoB (hyperapobetalipoproteinemia) and by a high ratio apoB/apoA-I. Later, C. Vergani and G. Bettale described a family with higher than normal prevalence of IHD and characterized by the deficiency of apoA-I (hypopalphalipoproteinemia). It was also observed that there are patients affected by premature coronary heart disease whose plasma levels of low-density-lipoprotein cholesterol are normal but who show a synthetic rate of apoB that is significantly higher than normal. How the high flux of apoB may be atherogenic is still unclear. A significant understanding of the clinical role of apolipoproteins, however, has been achieved.